EXHIBIT 605

FORENSIC MEDICAL

May 17, 2011

Mr. Matthew Moriarty
Tucker Ellis & West, LLP
1150 Huntington Bldg.
925 Euclid Avenue
Cleveland, Ohio 44115

Re: McCornack v. Actavis Totowa, LLC

Dear Mr. Moriarty:

This letter serves as my expert medical opinions regarding the death of Mr. Daniel McCornack. I am currently the Chief Medical Examiner of Nashville/Davidson County in Nashville, Tennessee. I also serve as the Chief Medical Officer of Forensic Medical Management Services. I practice forensic pathology exclusively. I am board certified by the American Board of Pathology in anatomic, clinical, and forensic pathology. In addition to my primary medical examiner practice, I also provide expert medical opinions in forensic pathology as a consultative service. This consultative service consists of less than 10% of my professional practice I hold medical licenses in Tennessee and Mississippi. I do not keep a list of cases in which I have provided expert medical opinions or testimony.

In forming my opinions in this matter, I have reviewed the following material: medical records from Dr. Gordon Lemm from 1994 through 2008; various laboratory reports from different sources dated 1994 through 2007; radiograph reports from various sources dated 1995 through 2005; pathology reports from skin and colon biopsies; undated electrocardiograms; medical records from Dr. Lawrence Von Dollen dated 1998 through 2007; additional laboratory reports dated 2002 through 2007; cardiology consultation from Dr. Roger Winkle; echocardiography reports dated 1995, 2001, and 2006; electrocardiograms dated 2000, 2001, 2004, 2006, and 2007; death certificate issued April 7, 2008; autopsy report; amended death certificate dated 9/30/2009; autopsy report (presumably amended) with revised cause of death, manner of death, and pathologic diagnoses; laboratory reports from NMS Labs dated June 24, 2008, May 29, 2009, and September 22, 2009; CVS Caremark recall letter; FDA statement of recall for Digitek® tablets; Santa Cruz County Sheriff-Coroner report; opinion of Keith Patrick Gibson, Pharm.D., J.D.; and depositions of Kathy McCornack, Dr. Von Dollen, Matthew McMullin, Dr. Gordon Lemm, and Dr. Richard Mason.

Mr. McCornack was a 45 year old white male found unresponsive by his wife near midnight on March 22, 2008 during a camping trip. Emergency medical personnel responded and Mr. McCornack was pronounced dead a short time later at 00:52 on March 23, 2008. He was apparently in his normal state of health and was not ill prior to being found unresponsive. His past medical history is significant for atrial fibrillation, hypertension, obesity, and hypercholesterolemia. An autopsy was performed by

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Dr. Richard Mason on March 26, 2008, approximately 79 hours after death. Blood was submitted to NMS Labs for toxicology testing. Dr. Mason certified the cause of death as ventricular arrhythmia due to atrial fibrillation due to hypertensive and atherosclerotic cardiovascular disease with exogenous obesity as a contributory cause. The manner of death was certified as natural. However, an undated amended autopsy report inexplicably changes the cause of death to ventricular arrhythmia due to digoxin toxicity due to digoxin poisoning. In addition, an amended death certificate dated September 30, 2009 also lists digoxin toxicity due to digoxin poisoning as the cause of death with hypertensive and atherosclerotic cardiovascular disease and exogenous obesity as contributory causes.

After review of all available information, it is my expert medical opinion that Mr. McCornack died of hypertensive and atherosclerotic cardiovascular disease with obesity as a contributory cause. There is no medical or scientific basis to support the diagnosis of digoxin toxicity or digoxin poisoning as causing, or in any way contributing to, Mr. McCornack's death. As documented in the autopsy report, Mr. McCornack has several cardiac findings capable of causing sudden cardiac death: enlarged heart (500 grams), left ventricular hypertrophy, coronary atherosclerosis, and myocardial fibrosis. The enlarged heart and thick left ventricle are due to long-standing hypertension. The myocardial fibrosis is indicative of previous myocardial ischemia or previous myocarditis. Any of these three conditions are well-documented causes of sudden cardiac death by causing a fatal arrhythmia, typically a ventricular arrhythmia. Obesity is a contributory cause of death. Additional autopsy findings include pulmonary congestion and edema and fatty liver. Pulmonary congestion and edema are non-specific findings. The fatty liver is most likely secondary to obesity; however, without microscopic sections, other etiologies cannot be completely excluded.

NMS toxicology report dated June 24, 2008 for postmortem blood of Mr. McCornack provides the following results: 0.048% blood ethanol, 630 ng/mL diltiazem, 3.6 ng/mL digoxin, trace quinine, and atropine. The ethanol in the blood may be due to antemortem alcohol ingestion, from postmortem in vivo production, or from a combination of both. Atropine is from attempted resuscitation. The source of the trace quinine is unknown. Diltiazem and digoxin were both prescribed for management of Mr. McCornack's cardiac conditions and are expected findings in the postmortem toxicology. As described below, the diltiazem and digoxin levels are non-toxic and would not contribute to death.

Due to postmortem redistribution, the levels of diltiazem and digoxin reported in the blood do not accurately reflect their levels in Mr. McCornack's blood when he was alive. Reliance on the postmortem digoxin level alone to determine the cause of death has no scientific or medical basis. Therefore, Dr. Mason's revised opinion that Mr. McCornack died of digoxin toxicity is erroneous and lacks scientific foundation. Postmortem redistribution describes the substantial changes that can occur in drug concentrations in the blood between death and when a postmortem blood sample is drawn (typically at autopsy). The process of postmortem redistribution is well recognized in forensic pathology and is abundantly documented in the forensic scientific literature. It generally refers to the diffusion of a drug across a concentration gradient in the body after death, but it also refers to the movement of drugs that are highly bound in specific types of tissue (such as digoxin in heart muscle) into other body compartments. Postmortem distribution can begin shortly after death and will continue as the postmortem interval increases. Redistribution, although diminished, will even occur in blood drawn from peripheral vessels. In fact, it has been determined that serum digoxin levels nearly always increase after death due to leaching from the muscle, with an average antemortem/postmortem ratio ranging from 1.42 for femoral vein blood specimens to 1 96 for heart blood specimens (Disposition of Toxic Drugs and Chemicals in Man, Eighth Edition, Baselt, 2008). Since the postmortem blood from Mr. McCornack was drawn from the axilla, there is a great potential for an increased axillary vein digoxin

level due to cardiac muscle leaching. Animal studies support the finding that "antemortem digoxin levels cannot be reliably inferred on the basis of high postmortem levels of the drug alone" (Ferner, RE. *British Journal of Pharmacology* 2008; 66:4, 430-443). Therefore, the 3.6 ng/ml of digoxin as reported by NMS in postmortem blood is not an accurate reflection of the antemortem digoxin level in Mr. McCornack, and the antemortem level was lower than the postmortem level. While some patients may manifest digoxin toxicity with "normal" levels of digoxin in the blood, the clinical diagnosis relies on signs and symptoms that, as discussed below, were simply not present in Mr. McCornack. In short, this postmortem digoxin level alone cannot be used as a basis for the diagnosis of digoxin toxicity.

The circumstances and investigative information surrounding someone's death must be taken into consideration with the autopsy findings and toxicology results when forming a final opinion regarding cause of death and manner of death. Not only is the digoxin level alone not high enough to cause toxicity and death, none of the symptoms typically reported with digoxin toxicity (nausea, vomiting, dizziness, blurred vision, decreased consciousness) were reported by Mr. McCornack just prior to his death. According to the Santa Cruz Sheriff-Coroner report Mr. McCornack was described as never being in any discomfort or pain that day. His wife said he did not complain of vomiting, vision changes, or any irregular heartbeats on the day of his death.

Finally, evaluation of digoxin pills submitted to NMS for examination shows that they are all of relatively uniform strength, weight, and thickness.

I also disagree with and find no medical basis for the final opinions expressed by Keith Patrick Gibson, Pharm.D., J.D. First, Mr. Gibson is simply not qualified, either in education or experience, to offer an expert medical opinion of cause of death. Second, his opinion that "Mr. McCornack had an elevated digoxin level at the time of his demise" is directly contradictory to other evidence and facts contained in Mr. Gibson's own report. And, third, Mr. Gibson's statement that "the elevated digoxin level was probably the result of a change in formulation of the Digitek tablet or a non-conforming tablet" is speculative, at best.

In summary, after review of all available information, in my expert medical opinion, Mr. McCornack's cause of death is hypertensive and atherosclerotic cardiovascular disease, with obesity as a contributory cause. The manner of death is natural. Digoxin toxicity did not contribute to or cause Mr. McCornack's death.

Sincerely,

Dr. Amy R. McMaster